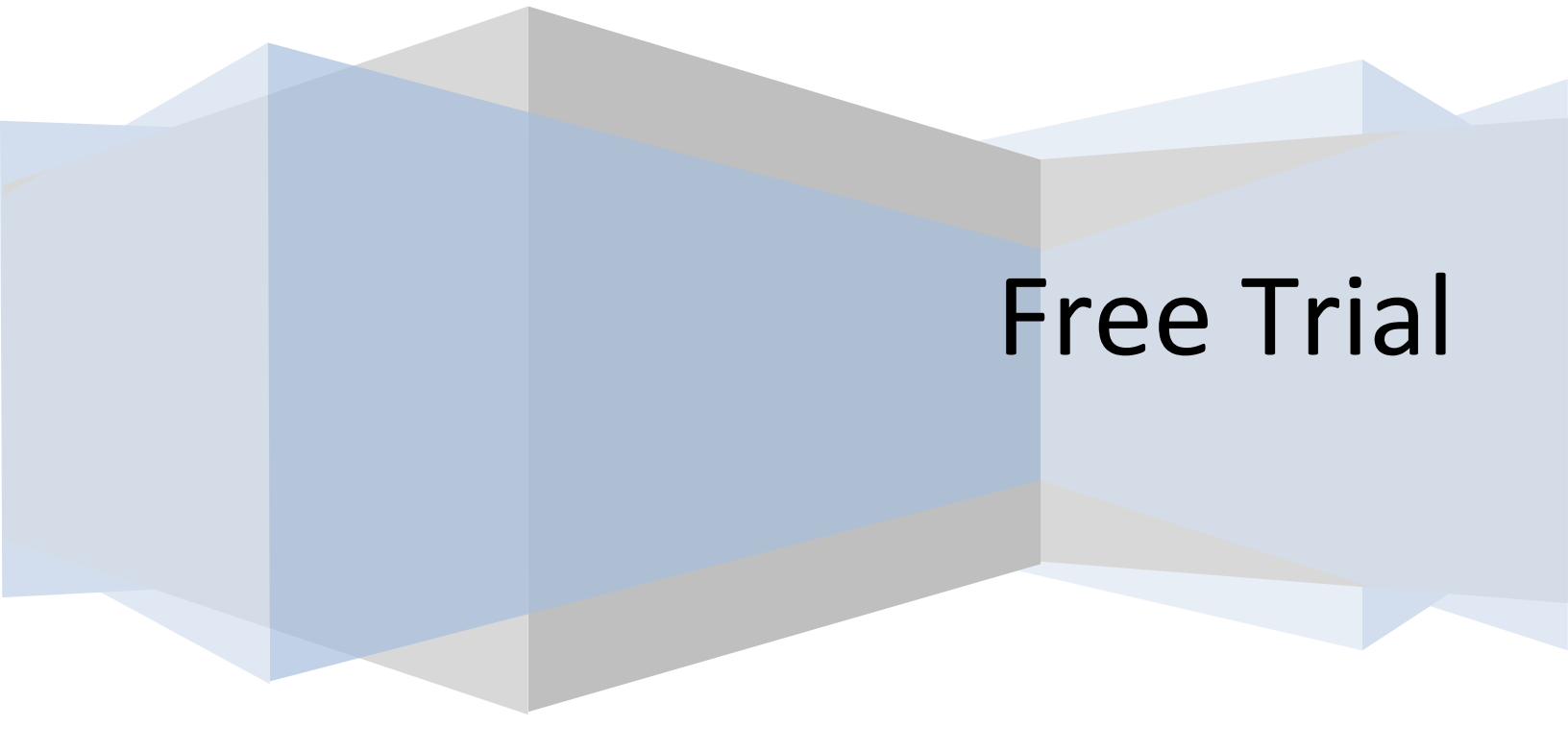


Dermatology

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Superficial Fungal Infections

1- Dermatophytoses

Epidermomycosis	Trichomycosis	Onychomycosis
Dermatophytosis of epidermis	Dermatophytosis of hair & hair follicle	Dermatophytosis of nail apparatus
Tinea Corporis	Tinea Capitis	Tinea Unguium
Caused by: Trichophyton rubrum (is most frequent) Risk factors: Typically seen in hot, humid climates Lesion: - Large, scaling, well-demarcated plaques (Single or scattered multiple lesions). Peripheral enlargement & central clearing produce → crinate pattern with concentric rings lesions.  Diagnosis: clinically + a skin scraping & KOH examination → microscope: Dermatophytes are recognized as septated, tubelike structures (hyphae) Treatment: Topical antifungals is preferred treatment	Caused by: T. tonsurans (90%) Lesions: 1- Non-inflammatory lesions: A) Scaly : - One, round, fine scaly sharply marginated "grey patch" with partial alopecia (off-broken hairs)  B) Black dots: - Broken-off hairs at scalp surface give appearance of "dots" in dark-haired patients within alopecic areas.  2- Inflammatory lesions: C) Kerion: - Extremely tender boggy, purulent, inflamed nodules & plaques Treatment: Systemic antifungals with Griseofulvin	Caused by: Trichophyton rubrum Lesions: 1- Distal subungual onychomycosis : Presents as onycholysis, subungual debris and discoloration beginning at the hyponychium that spreads proximally. 2- Proximal subungual onychomycosis : Begins under underneath the proximal nail fold.  Treatment: Systemic antifungals

2- Candidiasis

Etiology: <i>Candida albicans</i>. An oval yeast Ecology Candidiasis is usually an endogenous infection. <i>C. albicans</i> & other species are a commensal organism in the mouth & GIT → it causes opportunistic infection. KOH examination: Budding yeast forms AND sausage-like pseudohyphal forms.			Predisposing factors - Immunocompromise - Diabetes mellitus - Obesity - Hyperhidrosis, heat, maceration - Systemic & topical glucocorticoids - Chronic debilitation. - Pregnancy. - Moist opposing skin folds.		
Cutaneous candidiasis	Chronic candidal paronychia	Mucosal candidiasis			
1- Intertrigo (Erythema. Pruritus, tenderness, pain.) - Increased temp. & moisture in cutaneous folds make them susceptible. 2- Interdigital Most common in obese elderly. Distribution : - Hands: usually between 3rd & 4th fingers. - Feet: maceration in webspace 3- Diaper Dermatitis - Erythema, edema with papular and pustular lesions; erosions, oozing, collarette-like scaling at the margins of lesions Treatment: Topical antifungal agents: Nystatin cream : Effective for <i>Candida</i> only Imidazole creams Effective for candidiasis, dermatophytosis, pityriasis versicolor. Oral antifungal agents: Azoles (fluconazole & itraconazole): treat cutaneous infection. Nystatin : Eradicates bowel colonization.	Usually seen in wet workers & house wives. Nail fold: boggy, swollen, erythematous, tender on pressure. Nail plate may become irregular & discolored. 	1- Oral Candidiasis (thrush) : Adherent white patches on the tongue and inner surface of cheeks, if scraped off a raw bleeding area will be revealed. - Seen with prolonged use of antibiotics or corticosteroids. - It's maybe painful & interfere with eating.  2- Genital : a- Vulvovaginitis : Erythematous inflamed mucous membrane with white adherent plaque associated with itching & white creamy thick discharge. b- Monilial balanitis : Tiny white pustule or papules with soreness and irritation.			

3- Pityriasis Versicolor

Etiology: *Malassezia globosa* → Lipophilic yeast that **normally resides** in the keratin of skin & hair follicles (**An opportunistic organism**)

Predisposing Factors:

- Warm season or climates; tropical climate.
- Hyperhidrosis; aerobic exercise.
- Glucocorticoid treatment. • Immunodeficiency.

Skin lesions:

- **Macules** which may be confluent together forming **patches**, sharply margined, varying in size.
- **Fine scaling** is best seen by gently scraping of the lesions with the edge of glass slide.
- **Color:** 1- Hypopigmented 2- Hyperpigmented

Investigations:

1) **Direct Microscopic:**

- **Examination of Scales Prepared with KOH:** Filamentous **hyphae & globose** yeast forms (termed **spaghetti & meatballs**), are seen.

2) **Wood Lamp:**

- Blue-green fluorescence of scales

Treatment (Topical):

Selenium sulfide "lotion or shampoo"

Azole creams (ketoconazole) Apply daily or twice daily for 2 weeks

Terbinafine :1% solution Apply twice daily for 7 days

Tinea versicolor (pityriasis versicolor)

Pathogenesis

Malassezia globosa skin flora grows in exposure to hot & humid weather

Clinical features

- Hypopigmented, hyperpigmented, or mildly erythematous lesions (face in children, trunk & upper extremities in adolescents & adults)
- +/- Fine scale
- +/- Pruritus

Diagnosis

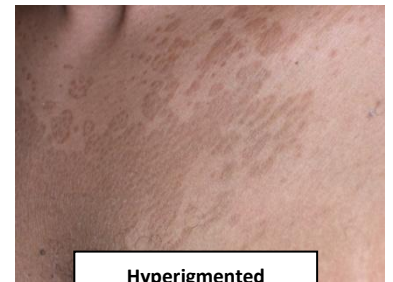
KOH preparation shows hyphae & yeast cells in a "spaghetti & meatballs" pattern

Treatment

Topical ketoconazole, terbinafine, or selenium sulfide



Hypopigmented



Hyperpigmented

Pityriasis rosea

- Acute **exanthematous eruption** with a **distinctive morphology** and often with **characteristic self-limited course**.

- **Caused by:** reactivation of HHV-7 or HHV-6, two closely related β -herpesviruses

Morphology:

- 1- **Initial lesion:** "**Herald**" **patch** develops usually on the trunk (Oval, slightly raised **plaque** or **patch** 2–5 cm, salmon-red color)
- 2- **Followed by:** 1 or 2 weeks later a **generalized eruption** develops in a typical distribution pattern ("**Christmas tree**" pattern)
- 3- **Spontaneous remission** in 6–12 weeks or less. (Recurrences are uncommon)



Herald patch



Christmas tree

Scarlet Fever

- **Scarlet fever** is caused by strains of **Group A streptococcus** that produce **erythrogenic exotoxins**.
- **Mode of transmission & age of distribution:** as streptococcal pharyngitis:

- Illness may follow a streptococcal pharyngitis, wound infections, burns, or streptococcal skin infection.

- Incubation period of **1 to 7 days**

→ followed by the illness **acutely** with Initial symptoms include: **fever, chills, toxicity + abdominal pain & pharyngitis**

→ within **12 to 48 hours:** **rash** initially appears on the **neck, axillae, and groin**,

→ Subsequently **generalizes within 24 hours**.

- **Rash:** - Punctate or finely Papular texture hence, the "**sandpaper-like**" description.
- Rash is blanchable on pressure.
- Rash fades by day 5-6 but residual petechial lesions are seen in cubital fossa (**Pastia sign**)
- **Pharynx** is typically **erythematous, swollen** and possibly covered with **gray-white exudates**.
- Strawberry tongue** is also a feature
- **Circumoral pallor:** area around mouth appears pale in comparison with extremely red cheeks

Towards the end of the first week, **desquamation (from face down to trunk finally to hands & feet)**

- **Treatment** is **Penicillin V (drug of choice)**

Erythromycin, Clindamycin, 1st generation Cephalosporins are good alternatives

Exanthema scarlet fever



Scarlet fever



Other Childhood Exanthematous Rashes

Measels (Rubeola)	Rubella (German measles)	Exanthema subitum (Roseola/HHV-6)	Erythema infectiosum (parvovirus B19)
Cause: Rubeola virus (RNA Paramyxovirus)	Cause: German measles	Cause: HHV-6	Cause: parvovirus B19
Prodrome: after 10 days 1- High grade fever/anorexia with: 2- Non-productive Cough (Bronchitis) 3- Coryza (Rhinitis) 4- Non-purulent Conjunctivitis 5- Koplik's spots (pathognomonic)	Prodrome: after 14-21 days Low grade fever with : 1- Conjunctivitis 2- Coryza 3- Cervical Lymphadenopathy 4- Forschheimer spots (rose spots appear on soft palate before rash)	Prodrome: Abrupt High grade fever for 3-4 days	Prodrome: Very mild , or NO fever
Pathognomonic: Koplik's spots Bluish-white lesions appear on erythematous buccal mucous membranes, - opposite to 1st & 2nd upper molars - sometimes on - inner conjunctivae & - vaginal mucosa	Pathognomonic: Tender and enlarged : 1- Occipital LNs 2- Posterior-auricular LNs <i>Posterior cervical & posterior auricular lymphadenopathies are common</i>		
Exanthem : 1- Blanching reddish-brown maculopapular rash 2- Cephalocaudal & centrifugal spread : Starts on the face behind ears, 24 hours then to → neck → trunk → arms → lower limbs Spare the palms & soles	Exanthem : appears 1-5 days later 1- Cephalocaudal spread of blanching erythematous maculopapular rash (starts on face & then spreads to trunk & limbs) - Rash is lasting for less than 3 days In adults: Same as children + Polyarthralgias	Exanthem : 1- Fever subsides on 5th day then: 2- Centrifugal spread of maculopapular rash (starts on trunk and then spread to extremities sparing the face!)	Exanthem : Sudden appearance of "Slapped Cheeks" rash : - Maculopapular - Pruritic - Worsen with fever & sun Exposure Starts on arms and spreading to trunk and legs.
Diagnosis: mainly clinical 1- PCR 2- Serology for anti-measles IgM & IgG → 4 fold increase in titres 3- Lab findings include : leukopenia & lymphopenia	Diagnosis: mainly clinical 1- PCR 2- Serology for: anti-rubella IgM & IgG		
Complications : 1- Otitis Media 2- Pneumonia 2- Neurologic : - Encephalitis - Acute disseminated encephalomyelitis (within weeks) - Subacute sclerosing panencephalitis (within years) 3- Gastroenteritis	Complications : 1- Encephalitis 2- Thrombocytopenia 3- Congenital rubella syndrome: (Risk is high in 1st trimester) - Sensorineural hearing loss - Intellectual disability - Cardiac anomalies (PDA) - Cataract - Glucoma	Complications : Most common cause of febrile fits from rapid fever onset	Complications : 1- Arthropathy 2- Aplastic crisis in SCC in HS patient
Prevention : - Live attenuated measles vaccine Treatment : 1- Supportive care 2- Vitamin A for hospitalized children - Vitamin A has been shown to: a- reduce the morbidity & mortality rates of patients with measles through immune enhancement. b- It also helps the gastrointestinal and respiratory epithelium to regenerate.	Prevention : - Live attenuated rubella vaccine Treatment : 1- Supportive care: Acetaminophen Patients can be infectious from 1 week prior to the onset of rash to 15 days after		
			

Varicella :

Herpes zoster (shingles) is the reactivation of VZV and occurs in dermatomal distribution

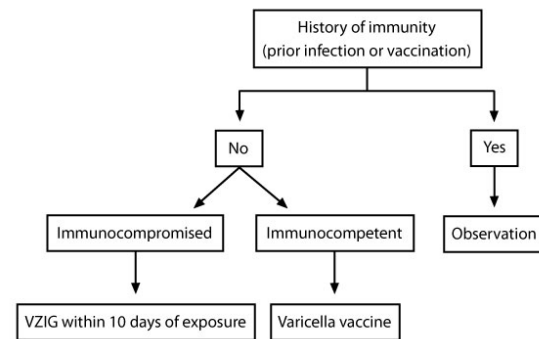
- Highly contagious, self-limited viral infection **caused by: Varicella-zoster virus (VZV)**, group of herpesviruses.
- Often, there is a history of exposure to infected individual, 90% of patient < 10 years old child.
- Prodrome:** - Fever/Anorexia/Headache/malaise/ abdominal pain before the onset of the rash.
- Rash:**
 - Erythematous macules, papules, vesicles**, scabbed lesions are **present at the same time** → Within days, vesicles become turbid and then crusted.
 - There is **centripetal distribution**: First **on face** and spread to **trunk** and **extremities**, (**sparing palms & soles**)
- Diagnosis:** is mainly clinical: PCR test of swab from the vesicles.
- Complications :**
 - Skin lesions may be superinfected by bacteria (Streptococcus pyogenes or Staphylococcus aureus).
 - Pneumonia in immunocompromised or pregnant patients.
 - Encephalitis.
 - Reye syndrome** (associated with aspirin use)
- Treatment :**
 - For most immunocompetent children: **Symptomatic for fever and pruritus**
 - For VZV pneumonia and for immunocompromised individuals: **Acyclovir**



Post-exposure prophylaxis :

- Patients age **more than 1 year** who are:
 - Nonimmune**
 - Asymptomatic**
 - Immunocompetent**
 → should receive the **varicella vaccine** for post-exposure prophylaxis **in < 5 days**.
 - Vaccine is **70%-100% effective** in preventing infection **if given within 3-5d. of exposure**
- Patients who are:
 - Nonimmune**
 - Immunocompromised**
 → should receive **passive with varicella zoster immunoglobulin (VZIG)** within **10 days of exposure** as they are at risk for life-threatening complications → VZIG may not prevent infection BUT it can reduce severity → So, require close monitoring for development of varicella infection as VZIG can **prolong the incubation period beyond 1 month**.

Varicella post-exposure prophylaxis



- Because the varicella vaccine is a live attenuated virus preparation, it is **contraindicated** in : 1- **Pregnant women** 2- **Immunocompromised** hosts.

Rosacea :

- Age:** most commonly occurs in **30- to 60-year-old patients** with fair skin, light hair and light eye color.
- Pathogenesis** is not known, although hair follicle mites have been thought to play a role.
- It's **chronic skin infection** that is characterized by: a **rosy hue with telangiectasia over cheeks, nose, chin**
Sometimes, **Papules** and **Pustules** may be present
- Precipitations:** Flushing of these areas is typically precipitated by:
 - Hot drinks**
 - Heat**
 - Emotion**
 - other causes of rapid body temperature changes.**
- Course:** The episodes are usually **intermittent**, but can **progressively lead to permanently flushed skin**.
- Treatment:** 1) **Medical:** is aimed at the inflammatory papules, pustules, and erythema.
2) **Laser surgery:** for telangiectasias



Henoch-Schonlein purpura

- A common **vasculitic** condition of childhood.
- It is commonly seen **after an upper respiratory tract infection** / and more common in males.
- The classic findings are :
 - Palpable purpura** in lower extremities & buttocks.
 - Renal findings:** hematuria & proteinuria.
 - Arthralgias:** most commonly affect the **knees & ankles**.
 - When patients present with **abdominal pain**, the two common pathologies which should be ruled out emergently are: 1- **GI bleeding**
2- **Intussusception**



—These symptoms are **always transient**, and there is **no** permanent damage to the joints

- Treatment** includes: 1- **administration of steroids**
2- **monitoring of renal function.**

Henoch-Schönlein purpura	
Pathogenesis	IgA-mediated leukocytoclastic vasculitis
Clinical manifestations	- Palpable purpura - Arthritis/arthralgia - Abdominal pain, intussusceptions - Renal disease similar to IgA nephropathy
Laboratory findings	- Normal platelet count & coagulation studies - Normal to ↑ creatinine - Hematuria +/- RBC casts +/- proteinuria
Treatment	- Supportive (hydration & NSAIDs) for most patients - Hospitalization & systemic glucocorticoids in patients with severe symptoms

RBC = red blood cell; NSAIDs = nonsteroidal antiinflammatory drugs.

Bacterial Infections

1- Superficial epidermal Infections

Intertrigo

- Nonspecific inflammation of opposed skin " intertriginous areas"
(inframammary regions, axillae, groins, gluteal folds)
- **Predisposition:** high surface humidity, trauma as friction , skin diseases
- **Clinical findings:** Erythema ± symptoms of pruritus, tenderness, or increased sensitivity
- **Causes :**
 - Bacteria :
Group A streptococcus, group B streptococcus, *C. minutissimum* (Erythrasma) , *P. aeruginosa*



2- Soft tissue infections

Cellulitis

- Acute infection of **Dermis & Deep subcutaneous tissue**
- **Etiology:** 1- **Staphylococcal aureus** &
2- **Group A beta-hemolytic Streptococci**
are the most common agents.
- **Clinical Features:**
 - 1- **Systemic signs :**
 - High-grade fever with rigors & malaise, fatigue, confusion
 - 2- **Local signs:**
 - Generalized swelling which is **erythematous, warm, tender,**
less well-demarcated than erysipelas

How can you differentiate erysipelas from cellulitis?

Cellulitis is characterised by:

- Lesions are primarily **NOT raised.**
- **Ill-defined border**

- **Necrotic changes** in deeper tissues are **NOT seen.**



Erysipelas

- Acute **superficial** infection of **Dermis & upper S.C. tissue.**
- **Etiology:** **Group A beta-hemolytic Streptococci (GAS)**
(Organism enter the skin through **abrasions**)
- **Clinical Features:**
 - 1- **Local signs:**
 - **Erythematous, warm, tender , oedematous, indurated ,**
**+
Raised
Sharply-demarcated PLAQUE**
 - In severe cases, overlying epidermis may show:
Bullae, Pustules or **Necrosis.**
 - It usually occurs **over the cheek**
 - There often a **history of trauma** or pharyngitis
 - 2- **Lymphatic involvement:**
 - Overlying **skin streaking & regional lymphadenopathy** indicate lymphatic involvement.



- **Treatment :**
 - 1- When systemic signs are present : IV **Nafcillin or Cefazolin**
 - 2- In areas with high prevalence of MRSA: **Vancomycin**

- **Treatment :**
Over 80% of the cases are due to **Streptococci**;
thus, → **Penicillin** is the drug of choice

Necrotizing fasciitis (History of **diabetes mellitus** should be suspected in patients with necrotizing fasciitis)

- It is a **rapidly spreading** infection involving the **fascia of deep muscles.**
- **Causes:** 1- It usually occurs **after trauma** 2- It may also occur **around foreign bodies** (in surgical wounds).
3- it can be **idiopathic** (e.g., scrotal or penile necrotizing fasciitis).
- **Organisms:** **Group A hemolytic Streptococci & Staphylococcus aureus** are frequently the initiating infectious bacteria; (it may other aerobic & anaerobic).
- **Clinical features :**
 - **Signs of systemic toxicity** (e.g., fever, hypotension) may be present.
 - There is a **sudden onset of pain and swelling at the site of trauma or recent surgery.** → progresses to **purplish discoloration & gangrenous changes.**
 - A gloved finger can easily be passed between the 2 layers, revealing **yellowish green necrotic fascia**, which helps in the diagnosis.
- **Investigations:** **CT scan** is useful in identifying the involved site, which reveals: 1- **necrosis** 2- **asymmetrical fascial thickening** 3- **gas in the tissues.**
- **Treatment :**
 - 1- **Thorough surgical debridement** of all the necrotic tissues is the most important therapy.
 - 2- **High-flow oxygen, fluid resuscitation, and broad-spectrum antibiotics** should be included in the management.

3- Pyoderma

Impetigo

- A Superficial pyogenic skin infection :
 - Infections of the **epidermis (impetigo)**
 - which may extend into the **dermis (ecthyma)**

Portal of entry

- **Primary infections** in minor superficial breaks in the skin
- **Secondary infections** of preexisting dermatoses (impetiginization or 2ry infection)

Age of Onset :

- **Primary infections:** more common in children
- **Secondary infections,** at any age.
- **Bullous impetigo:** **neonates, especially; children <5 years old.**

Etiology:

- either or both :
 - 1- **Staphylococcal aureus** (most commonly)
 - 2- **Group A beta-hemolytic Streptococci**

In bullous impetigo: **epidermolytic toxin A- (etA) gene producing S. aureus** which also causes **Staphylococcal Scalded Skin Syndrome (SSSS).**

Types:

1- Vesiculo-Pustular (Non-bullous) impetigo :

Etiology:

- either or both :
 - 1- **Staphylococcal aureus** (most commonly)
 - 2- **Group A beta-hemolytic Streptococci**

Complications: may **post-streptococcal glomerulonephritis.**

Predisposing factors :

- Warm and humid climate - poverty - crowding
- Poor personal hygiene - Carriage of GABS or S. aureus.
- **Nasal carriage of Staphylococci** can cause **recurrent impetigo.**

Clinical features :

- Presents as **erythematous macule**, → which rapidly evolves into **vesicles & pustules** → pustules later rupture and leave: **honey-colored, crusted exudates.**

- **History of** 1- **skin trauma** or 2- **insect bite** is common
- **Local lymphadenopathy** can present.

Treatment :

- **Topical mupirocin** is the treatment of choice
- Alternatives: 1- **Oral Erythromycin**
- 2- **Oral Cephalexin / Dicloxacillin / Ampicillin** in severe cases.



Folliculitis

Definition:

Pyogenic infection of hair follicle with ± pus in the ostium of follicle.

Etiology: **Staphylococcal aureus** (MSSA, MRSA).

Classification:

- **Superficial** → **Acute:** Bockhart's impetigo **Chronic:** Acne keloidalis
- **Deep** → **Acute:** Furuncle/Carbuncle **Chronic:** Sycosis barbae – Pseudo folliculitis.

Pathogenesis: They represent a continuum of severity of *S. aureus* infection. → Portal of entry: hair follicle, break in the integrity of skin.

Furuncle

- **Initially:** Painful, solitary, erythematous, deep seated, follicular **NODULE.**
- Nodule **becomes** fluctuant, with **ABSCCESS** formation
- A variable zone of cellulitis may surround the furuncle.

Location :

- Mostly on:
 - 1- **Areas subjected to friction &**
 - 2- **Hair-bearing region**



Carbuncle

Evolution is similar to that of furuncle **BUT** : Composed of **several to multiple, adjacent, coalescing furuncles**



Sycosis barbae

- Discrete follicular **pustules**, each pierced by a hair, in the beard.
- Crustation follows, ending by scarring. New lesions develop as the old ones heal

Pseudo folliculitis

- Erythematous **papulo-pustules**, with skin-buried hair.
- It affects mainly the beard of blacks.
- It results from ingrowing hair, which predisposes to 2ry infection, caused mainly by *Staph. aureus*.



Staphylococcal Scalded Skin Syndrome (SSSS)

- **Age:**
 - SSSS is primarily a **disease of children** BUT adults with **renal disease or immunocompromise may also be affected**.
 - It is usually seen in **children less than 6 years of age**.
- **Caused by:** **exfoliative toxin-producing strains of Staphylococcal aureus** → The toxins **target desmoglein 1**, which is **responsible for keratinocyte adhesion in the superficial epidermis**.
- **Prodrome:** of 1- fever 2- irritability 3- skin tenderness
- **Eruption:** **Erythema starts on the face, and generalizes within the ensuing 24-48 hours** → **Superficial flaccid blisters** soon develop, with **flexural accentuation** and **perioral crusting**.
 - **Nikolsky sign is positive** (gentle lateral pressure on the skin surface adjacent to a blister causes slipping & detachment of a superficial layer of skin)
 - The blisters of SSSS are **fragile and when unroofed reveal a moist erythematous base**.
 - Subsequent **scaling & desquamation continue for about 5 days** and
 - the entire process usually **resolves within 1-2 weeks**.
- **Cultures** from **intact bullae** are usually sterile because this is a **toxin-mediated process**.
- **Treatment :**
 - 1- Eliminate any inciting focus of infection with **appropriate anti-staphy. antibiotics** and
 - 2- Provide supportive wound care of all denuded areas.



Felon :

- **Tailors** can develop **felon** due to **needle injuries**.
- **Differential Diagnosis:** It is important to distinguish felon from **Herptic whitlow**.
- **Clinical features:** Felon is a **bacterial infection of the distal volar space, characterized by a tense abscess and intense throbbing pain**.
- **Treatment:** **Incision & drainage with appropriate antibiotic (e.g., cephalosporins)** is the treatment of choice.

Systemic Inflammatory Response Syndrome (SIRS) :

- **Pathophysiology :**
 - There is usually an **original insult** (e.g., infection and injury) that leads to **inflammation & a dysregulated host response**, with a **massive and uncontrolled release of proinflammatory substances** causing **extensive tissue damage**. → This response to an infection is referred to as **sepsis**, while **noninfectious causes are known as systemic inflammatory response syndrome (SIRS)**.

Temperature >38.5°C (101.3°F) or <35°C (95°F)
Pulse >90/min
Respirations >20/min
WBC >12,000 cells/mm ³ , <4000 cells/mm ³ , or >10% bands

- **SIRS** is defined as having **at least 2 of the following 4 criteria**
- It can occur in : **pancreatitis, autoimmune disease, vasculitis , burns**.

Sepsis (i.e .. SIRS with a known infection) Criteria that indicate sepsis in the patients include:

- 1- **Worsening hyperglycemia** (due to worsening insulin resistance),
- 2- Leukocytosis,
- 3- Thrombocytopenia
- 4- Mild hypothermia (temperature <36C),
- 5- Tachypnea, and Tachycardia.

Sepsis is considered severe when there is **associated end-organ dysfunction, such as :**

- 1- Oliguria
- 2- Hypotension (i.e ..systolic <90 mm Hg)
- 3- Thrombocytopenia (i.e .. platelet count <80 ,000/mm')
- 4- Metabolic acidosis
- 5- Hypoxemia.

Complications of burns in 1st week:

- 1- Severe burns → manifest **some evidence of SIRS** → **sepsis & Septic shock** from **wound infections (S.s aureus or P.aeruginosa)**.
- 2- **Hypermetabolic response** : 1-Hyperglycemia (due to insulin resistance) 2- muscle wasting 3- protein loss 4- hyperthermia 5- Increased energy expenditure.

Viral Infections

1- Human Papilloma Virus (HPV) infections " Warts"

Clinical type	Site	Presentation	Serotype
Common (verruca vulgaris)	Hands	- Hyperkeratotic , scaly , rough, skin-coloured papules. - Characteristic “red or brown dots” are pathognomonic , representing thrombosed capillary loops. - painless – Usually multiple. 	2,4,29
Plantar (v. plantaris)	Soles against pressure points	- Early small, sharply margined papule → plaque with rough hyperkeratotic surface, - studded with brown-black dots (thrombosed capillaries). - Tender – single or multiple. 	1
Flat (v. plana)	Face & hands	- Small(2-5 mm), sharply defined, flat-topped, skin-colored or hyperpigmented papules. - They are multiple. 	3,10
Anogenital (Condyloma. Acuminatum)	Anogenital region	Moist, exophytic (cauliflower-like), papules/nodules, variably sized; - One of Sexually Transmitted Diseases. 	6,11

Treatment

Chemical	Common, Planter	Salicylic acid (Keratolytic) For small lesions 10–20% / For large lesions 40% for 1 week
	Flat	5-fluorouracil / Tretinoin
	External genital	Imiquimod cream / Podophyllotoxin
Surgery	All types	Cryosurgery : using cryospray, freezing the wart and 1–2 mm of surrounding normal tissue for approximately 30 s (Don't use in face as it cases scarring)
	Resistant	Electrosurgery: More effective than cryosurgery, but associated with scarring
	Resistant	Surgical Excision

2- Molluscum Contagiosum

- **Etiology:** Molluscum contagiosum virus (MCV), is **pox virus**.
- **Description :**
 - It's characterized by **multiple, dome-shaped lesions with central umbilication**.
 - Lesions typically have a **pink, translucent quality** and may be *arranged in a linear fashion* due to spread of the virus to adjacent
 - It can be **generalized in immunodeficient conditions** (such as AIDS, especially when CD4 count < 100/ul)
 - **Conjunctivitis** (as seen in this patient) may occur **if the lid margins have been infected**.
- **Duration of lesions :** Is it self limited ? YES, It usually persists up to **6 months** then undergo spontaneous regression.

Molluscum contagiosum



Molluscum contagiosum



3- Herpes Simplex Virus (HSV) infections

Etiology: **HSV-1, HSV-2.**

- **Labialis:** HSV-1 (80–90%), HSV-2 (10–20%).
- **Urogenital:** HSV-2 (70–90%), HSV-1 (10–30%).
- **Herpetic whitlow:** <20 years of age usually HSV-1
>20 years of age, usually HSV-2.
- Neonatal: HSV-2 (70%), HSV-1 (30%).

Treatment :

1) Topical Antiviral Therapy: Approved for herpes labialis.

Acyclovir ointment :

Approved for initial genital herpes & limited mucocutaneous HSV infections in immunocompromised individuals.

2) Oral Antiviral Therapy : Approved for **genital herpes & non-genital infections**.

- Antiviral agents more effective in treating primary infections than recurrences.
- *Most episodes of recurrent herpes do not benefit from oral acyclovir*

Clinical Manifestations of non-genital HSV infection

Primary Herpes

- **Incubation Period:** 2- to 20-day (average 6).
- Symptomatic primary herpes is **uncommon**.

Characterized by:

- **VESICLES** at the site of inoculation.
- Associated regional lymphadenopathy
- Constitutional symptoms :
fever, headache, malaise, myalgia. .

Mucocutaneous findings :

Skin findings:

- Sudden onset of grouped **VESICLES** on **erythematous base** which may evolve to **pustules**.
- They slough to form **erosions** which may enlarge to **ulcerations** which may be **crusted**.
- **Healing:** in 2–4 weeks, with resultant hypo or hyper-pigmentation, uncommonly with scarring.

Mucous Membranes

- **Oral mucosa** usually involved only in primary HSV infection at any site in the oropharynx.
- **Conjunctival & corneal** autoinoculation may occur.



Recurrent Herpes

- Prodrome of **tingling, itching** sensation **precedes visible skin changes by 24 h**.
- Constitutional symptoms are usually **absent**

Herpetic whitlow

- It is a common **viral infection of the hand** → **caused by** either **type 1 or 2 herpes simplex virus**, and is usually **self-limiting**.
- **Mode of transmission:** Direct inoculation of the virus through broken skin
- **Most commonly seen in** 1- **Women with genital herpes** or 2- **Children with herpetic gingivostomatitis**
2- **Health care workers** are also at **increased risk** of this infection, *due to contact with infected serum or saliva*.
- **Clinical presentation :**
 - **Systemic symptoms** such as fever and lymphadenopathy may occur
 - Patients often present with **throbbing pain in the distal pulp space, which is swollen, soft and possibly tender**.
 - **Lateral nail fold** may also be affected,
 - **Non-purulent vesicles on the volar aspects are clinically diagnostic**.
- **Diagnosis :** is confirmed by 1- **positive history of exposure** & 2- **multinucleated giant cells in the Tzanck smear of the vesicles**. This
- **Treatment:** It is a self-limiting illness → however: 1- **Oral acyclovir** & 2- **Topical bacitracin** to prevent secondary infection may be used.

4- Herpes Zoster Virus (HZV)

	Primary (Chickenpox)	Recurrent (Shingles)
Patients affected	More common in children	Patient with prior history of varicellazoster Infection
Timing of presentation	- Symptoms 21 wk after infection occurs; - Symptoms of headache, malaise, myalgias, and fever precede development of lesions by ,3 days	- Myalgias, fever, malaise preceding lesions by approximately 3 days
Type of lesion	Small, red macules that evolve into papules And then vesicles that eventually become crusted	Small, red macules that evolve into papules and then vesicles that eventually become crusted
Distribution of lesions	Wide distribution	- Limited to single or few distinct dermatomes ; - involvement of multiple dermatomes indicates disseminated disease
Course of disease	- Lesions may develop up to 1 wk and resolve a few days after appearing; infective until lesions crust over	- Lesions exist for a week and may be painful ; infective until lesions crust over
Treatment	1- Antipruritics aid symptoms; 2- Acyclovir used in - severe cases or - immunocompromised patients 3- vaccination has reduced disease incidence significantly	1- Analgesics, possible corticosteroids; 2- Acyclovir used in immunocompromised patients and trigeminal nerve distribution
Complications	More severe course in : 1. older and 2. pregnant patients (↑ risk of varicella pneumonia); can have severe consequences if passed from infected mother to unborn fetus	1- Post-infectious neuralgia (long-lasting pain at site of eruption): Treatment with antiviral agents decreases duration of symptoms & the incidence of post-herpetic neuralgia 2- Trigeminal neuropathy

Scabies

- **Scabies** is caused by an infestation of **Sarcoptes scabiei** → figure
- **Clinical features:** Patients complaints of **generalized itching**.
 - The characteristic lesions are **pruritic vesicles & pustules**
 - in **"runs" over the finger webs, heels of palms, and wrist creases**.
 - Pruritic papules may be seen over the **nipples & areola in females**, and **scrotum & penis in males**.
- **Examination** of scrapings from excoriated lesions under light microscopy reveals: **mites, ova, and feces**.
- **Treatment:**
 - For adults, the treatment is:
 - 1- **5% permethrin cream**, which is applied from the neck down and left overnight.
 - 2- **Low-potency topical steroids** can be added to treat the dermatitis.
 - 3- Bedding & clothing should be cleaned or set aside for 2 weeks.



Acne Vulgaris

- An inflammation of **pilosebaceous units**, very common
- Appears in **certain body areas (face, neck, trunk, back)**
- Most frequently in **adolescents**
 - Acne usually decreases in severity as adolescence ends.

Corticosteroid use and **androgen** production disorders are common causes of outbreaks in adulthood

Manifests as:

1- Comedones :

- Open comedones (blackheads)
- Closed comedones (whiteheads)

2- Papulopustules

3- Nodules

4- Cysts

- **Results in** → pitted, depressed, or hypertrophic scars

Acne Conglobata :

- **Severe cystic acne** with more involvement of the **trunk** than the face.
- Coalescing nodules, cysts, abscesses, ulceration and irregular scars , producing pronounced disfigurement.



Treatment of acne vulgaris



Comedonal acne

- Closed or open comedones on forehead, nose & chin
- May progress to inflammatory pustules or nodules
- Treatment: **Topical retinoids**; salicylic, azelaic, or glycolic acid



Inflammatory acne

- Inflamed papules (<5 mm) & pustules; erythema
- Treatment:
 - Mild: Topical retinoids + benzoyl peroxide
 - Moderate: Add topical **antibiotics** (eg, erythromycin, clindamycin)
 - Severe: Add oral antibiotics



Nodular (cystic) acne

- Large (>5 mm) nodules that can appear cystic
- Nodules may merge to form sinus tracts with possible scarring
- Treatment:
 - Moderate: Topical retinoid + benzoyl peroxide + topical antibiotics
 - Severe: Add oral antibiotics
 - Unresponsive severe: **Oral isotretinoin**

- It is important to assess severity of acne before beginning treatment:

1- Comedones ("blackheads" & "whiteheads") with minimal inflammation → represent mild disease :

- The best initial treatment is **topical retinoid**.

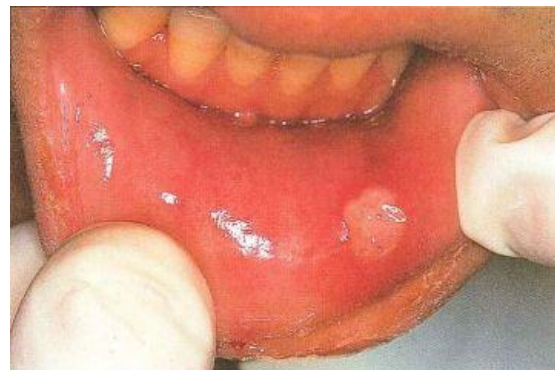
The effect of the treatment is usually slow, and patients should be made aware of this matter

2- Nodulocystic form & those who developed scar :

represent **moderate-to-severe** acne → Give **oral isotretinoin**

Aphthous ulcers

- They are described as **shallow, fibrin-coated ulcerations** with **underlying mononuclear infiltrates**.
- **Aphthae (canker sores)** are recurrent, self-limiting ulcerations of indeterminate (possibly autoimmune) etiology.
- **Site:**
 - These arise in the **mucosa of the oral cavity**, (**NOT found** in the **vermillion zone of the lips** or on the **gingiva**)
 - **NOT appear** in surfaces covered by **keratinized stratified squamous epithelium**.



Eczema / Dermatitis

1- Contact Dermatitis

Irritant Contact Dermatitis		Allergic Contact Dermatitis
- Most common form of occupational skin disease - It is caused by exposure of the skin to chemical or other physical agents that are capable of irritating the skin, acutely or chronically.		Caused by an antigen (allergen) that elicits: Type IV (cell-mediated or delayed) hypersensitivity reaction. <i>Host requires to be sensitized before a reaction will develop, so a patient must have had a prior exposure to the allergen before developing reaction.</i>
Dependent on concentration of the offending agent		Dependent on degree of sensitization: Minute amounts of the offending agents may elicit a reaction.
Occurs in everyone		Only in sensitized individuals
- Confined to the area of exposure → therefore always sharply margined & never spreads		- Reaction that tends to involve the surrounding skin (spreading phenomenon) and may even spread beyond affected sites.
Acute ICD	Chronic ICD	Allergic Contact Dermatitis due to Plants Termed: Allergic phytodermatitis (APD)
- Severe irritants cause toxic reactions after a short exposure. Skin findings : - occur minutes after exposure Evolution of Lesions Erythema with a dull, nonglistening surface → vesiculation (or blister formation) → erosion → crusting → shedding of crusts & scaling → necrosis → shedding of necrotic tissue → ulceration → healing. Duration : Days, weeks depending on tissue damage. Treatment : 1- Remove the etiologic agent 2- Topical class I glucocorticoid preparations. 3- In severe cases, systemic glucocorticoids may indicated.	- Most cases are caused by chronic cumulative exposure irritants. Cumulative ICD Skin findings : - Develops slowly after repeated additive exposure to mild irritants (water, soap, detergents, etc.), usually on hands. Evolution of Lesions Dryness → chapping → erythema → hyperkeratosis & scaling → fissures & crusting. Duration : Chronic, months to years. Treatment : 1- Remove the etiologic agent. 2- Employ a potent topical glucocorticoid preparation 3- provide adequate lubrication	• Cause: due to exposure to toxic plants (Toxicodendron dermatitis) such as: 1- Poison ivy (<i>Toxicodendron radicans</i>) 2- Posion oak 3- Poison sumac • Clinical features: ACD due to <u>poison ivy exposure</u> typically causes: - Rash arranged in a linear or streaked fashion because: <i>the allergen is typically inoculated onto the skin by the branches and leaves of the plant as the patient walks past it.</i> - The rash occurs 24-48 hours after exposure. - The erythematous streaks subsequently develop : edema, vesiculation and erosion with weeping drainage. <i>Vesicular fluid in contact dermatitis is usually sterile; however, the fluid may become secondarily infected (impetiginized) by streptococcal & staphylococcal organisms.</i> Pruritus is typically severe • Other common sensitizers : 1- Nickel (found in jewelry), 2- Formaldehyde (found in clothing and nail polish), 3- Certain fragrances, preservatives, rubber
 Acute irritant contact dermatitis on the hand due to an industrial solvent : There is massive blistering on palm.	 Early chronic irritant contact dermatitis in a housewife : This has resulted from repeated exposure to soaps and detergents. Note glistening fingertips (pulpitis).	 Linear vesicular lesions with erythema and edema on the calf at sites of direct contact of the skin 5 days after exposure with the poison ivy leaf.

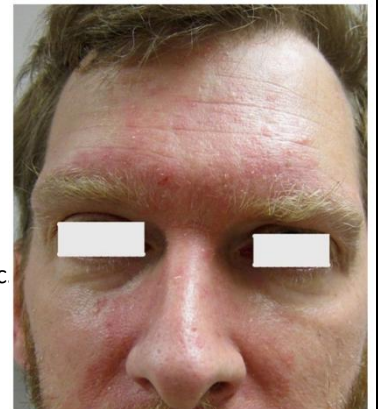
2- Atopic Dermatitis

- An acute, subacute, or chronic relapsing skin disorder
- **Age of onset:**
 - usually begins in **infancy** from 2 months – 1 year (in 60% of cases) → **Infantile AD**
 - begins by age of 5 years (in 30%)
 - begins from 6 years – 20 years (in 10%)
- **Risk factors:** History of atopy : 1- **Asthma** 2- **Allergic rhinitis** 3- **Family history**
- **Pathophysiology:**
 - Atopic dermatitis is the result of **decreased skin barrier function** due to improper synthesis of components of the epidermal **cornified cell envelope** → associated with 1- impaired filaggrin production, 2- reduced ceramide levels 3- increased transepidermal water loss
 - This allows allergens ready access to the deeper levels of the epidermis where they may generate the **immune response** characteristic of atopic dermatitis (**Type I (IgE-mediated) hypersensitivity reaction**)
- **Clinical features:** Atopic dermatitis in infancy:
 - **Distribution:**
 - 1- Typically affects : **face, scalp , extensor surfaces of the extremities** – a distribution differs significantly from that seen in **adults** : (mostly flexural surfaces)
 - 2- **The diaper region is typically spared.**
 - **Description :**
 - Lesions usually **begin with pruritus alone** and **evolve to erythematous excoriated Papules and Plaques** that may **weep** → **oozing erosions with cracked scaly skin** → And become **secondarily impetiginized.**
- **Treatment :**
 - 1- Treatment is with **improvement of skin barrier function** through 1- the use of mild cleansers & 2- thick, bland emollients
 - 2- in addition to **mild topical anti-inflammatory ointments** (topical corticosteroids or tacrolimus)
 - 3- **Severe cases:** treated with : 1- Oral corticosteroids 2- Anti-Histamines



3- Seborrheic Dermatitis

- **Chronic inflammatory papulosquamous disease** occurring in regions where the **sebaceous glands** are most active.
- **Age:** can affect all age groups → found with **increased frequency** in patients with :
 - 1- **Parkinson disease**
 - 2- **HIV** → severe intractable SD should be a clue to the existence of **HIV disease**
- **Pathogenesis:** *Malassezia furfur* is said to play a role in the pathogenesis, and response to **topical ketoconazole** & **selenium sulfide** is some indication.
- **Clinical features:**
 - **Distribution:**
 - 1- **Head :**
 - In infants : it often **begins on scalp** and called: **" cradle cap "** : **Erythema & yellow-orange scales & crusts on infants scalp.**
 - scalp (dandruff)
 - 2- **Face:** ("butterfly") areas
 - Areas most commonly affected : **eyebrows, nasolabial folds, bases of the eyelashes (blepharitis) and paranasal skin**
 - D.D: Erythema of SD is often overlooked and thought to be the flushing of rosacea. SD does not respond to ttt of rosacea*
 - Ears: **retroauricular, meatus.**
 - 3- **Trunk :**
 - **Tellowish-brown patches over the sternum common**
 - *D.D: Simulating lesions of pityriasis rosea or pityriasis versicolor*
 - 4- **Body folds :**
 - Axillae, groins, anogenital area, submammary areas, umbilicus, and diaper area
 - **Description :**
 - Transparent to **yellow papules** and occasional **greasy looking scaling plaques** are characteristic
- **Treatment :**
 - 1- Moisturizers
 - 2- Topical antifungals
 - 3- Anti-dandruff shampoos,
 - 4- Topical steroids. **Severe cases may suggest underlying immunodeficiency.**



Bullous Diseases

Pemphigus Vulgaris



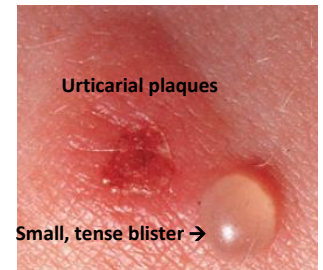
- Serious, acute or chronic, bullous autoimmune disease of **skin & mucous membranes**
- **Pathogenesis:**
 - Loss of the **normal cell-to-cell adhesion in the epidermis (acantholysis)** occurs as a result of circulating **Auto-Antibodies of the IgG class**; which bind to **desmogleins 3**.
- **Clinical manifestations :** → 2 types : Localized / Generalized
 - **Distribution:**
 - PV usually starts in the **oral mucosa** (rapidly become erosive & painful preventing eating), and months may elapse **before skin lesions occur**
 - Lesions may be **localized** for months, after which **generalized** bullae occur
 - **Description :**
 - It is characterized by **flaccid bullae**(blisters) that appear **spontaneously** → **when rupture** become **tender and painful** from erosions
 - **Extensive erosions that bleed easily** (second figure)
 - Easy separation of the epidermis on superficial pressure (**positive Nikolsky's sign**) is characteristic
- **Investigations :**
 - 1- Immunofluorescence microscopy reveals **IgG deposits intercellularly in the epidermis**
 - 2- IgG Autoantibodies to **intercellular substance of epidermis (IIF)**
 - 3- ELISA: Antibodies to **desmoglein 3** >> desmoglein 1
- **Treatment:** Immunosuppressive agents
 - Commonly used drugs for treatment are **steroids**.
 - **Azathioprine** may be used with **prednisone & methotrexate**.

Bullous Pemphigoid



Generalized eruption with confluent urticarial plaques and multiple tense blisters.
- The condition is **severely pruritic**.

- A bullous autoimmune disease usually in **elderly patients (60 – 80 years)**
- **Pathogenesis:**
 - Interaction of autoantibody with **bullous pemphigoid antigen [BPAG1 and BPAG2]** in **hemidesmosomes of basal keratinocytes** → is followed by complement activation and attraction of **neutrophils & eosinophils**.
- **Clinical manifestations :**
 - **Erythematous, Pruritic Papular or urticarial-type lesions** may precede **bullae formation** by months.
 - It is characterized by **tense blisters**, as opposed to the **flaccid blisters** in pemphigus.
 - Eruption may be **localized** or **generalized**
 - **Oral lesions are very rare**.
- **Investigations :**
 - 1- Immunofluorescence microscopy reveals **IgG & C3 deposits in the dermal epidermal junction**
 - 2- **Antibasement membrane IgG autoantibodies** in serum
- **Treatment:** Immunosuppressive agents
 - In very mild cases and for local recurrences, **topical glucocorticoid or topical tacrolimus**
 - **Systemic prednisone** wither alone or combined with **azathioprine**
 - **Low-dose MTX** is effective & safe in elderly

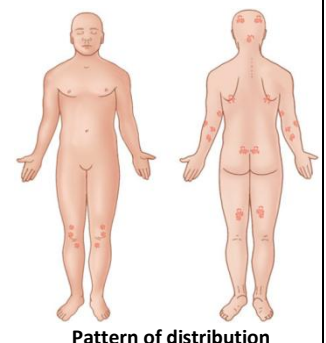


Dermatitis Herpetiformis



Generalized highly pruritic eruption with characteristic pattern of distribution

- A **chronic, recurrent, intensely pruritic eruption** occurring **symmetrically on the extremities and trunk**.
- **Clinical manifestations :**
 - **Description :**
 - It is manifested by **Pruritic Papules, vesicles & urticarial plaques**
 - **Typical distribution :**
 - Over the **elbows, knees, buttocks, posterior neck, and scalp**
- **Associated with** **gluten-sensitive enteropathy (GSE)**.
- **Investigations:**
 - 1- Immunofluorescence shows **granular IgA deposits along dermal papillae**.
 - 2- Circulating anti-endomysial antibodies can be detected in all patients.
- **Treatment :**
 - 1- **Gluten-free diet** &
 - 2- **Dapsone** are effective treatment options.



Pattern of distribution

Photo-Induced Disorders

Classification of Skin Reactions to Sunlight

1- Phototoxicity

Acute Sun Damage (Sunburn)	Phototoxic Drug Reaction	Plant-induced (phytophotodermatitis)
Sunburn is an acute, delayed, and transient inflammatory response of normal skin after exposure to UVR from sunlight or artificial source Sunburn reaction: Erythema, if severe: edema, vesicles and bullae; frequently resolves with hyperpigmentation Treatment : 1- Topical: - Cool wet dressings, - Topical glucocorticoids. 2- Systemic: Acetylsalicylic acid, NSAIDs. 	Adverse reaction of the skin that results from simultaneous exposure to certain drugs (via ingestion, IV, IM, or topical application) & to UVR Drugs cause phototoxicity: (FAST) Fluoroquinolones / Aminoglycosides, Sulfonamides / Tetracyclines Pathophysiology : Phototoxic drug reactions result from the production of reactive oxygen species by the interaction of drug metabolites with UVR. → These reactive oxygen products then directly damage cell membranes & DNA. - Prior sensitization is NOT required for phototoxic drug eruption; it is required for "photoallergic drug eruption" Clinical manifestations : - Patients present with an exaggerated sunburn reaction in a photo distribution; - Bullae may be present in a severe sunburn. → D.D. Typically the sun exposure time preceding development of the phototoxic eruption is less than would normally cause a sunburn in the patient.	Phytophotodermatitis (plant + light = dermatitis) is an inflammation of the skin caused by contact with certain plants during occupational exposure to sunlight. Pathophysiology : The inflammatory response is a phototoxic reaction to photosensitizing chemicals in several plant families. Commonly due to exposure to: 1- Limes 2- Celery 3- Meadow grass Clinical manifestations : - Acute: Erythema, edema, vesicles, and bullae - Often bizarre streaks, artificial patterns that indicate an "outside job".

2- Photoallergic

Chronic actinic dermatitis	Photo-allergic drug eruption	Solar urticaria
"Photoallergic dermatitis" can persist for months to years. This is known as : Persistent Light Reaction or (Chronic actinic dermatitis) Clinical manifestations : The condition persists despite discontinuation of the causative photoallergen, with each new UV exposure aggravating the condition. Chronic eczema-like lichenified and extremely itchy confluent plaques result	This results from interaction of a photoallergen and UVA radiation. - In sensitized individuals exposure to a photoallergen and sunlight results in a pruritic eczematous eruption confined to exposed sites and clinically indistinguishable from allergic contact dermatitis (D.D.) - In most patients the eliciting drug has been applied topically, BUT systemic elicitation also. Diagnosis : History + Clinical + requires the use of patch and photopatch tests.	Uncommon sunlight-induced whealing confined to exposed body sites. - Eruption occurs within minutes of exposure and resolves in a few hours. - It's an immediate type I hypersensitivity response to cutaneous and/or circulating photoallergens. Treatment: 1- Multiple phototherapy sessions 2- Oral immunosuppressive agents or 3- plasmapheresis.

3- Metabolic & Nutritional

Porphyria cutanea tarda	Pellagra
It's a condition that arises from the deficiency of: (acquired or hereditary) uroporphyrinogen decarboxylase, enzyme in the heme synthesis pathway. It can be triggered by: the ingestion of certain substances : (e.g .. ethanol, estrogens) OCPs, that should be discontinued once suspected. It is often associated with: Hepatitis C infection. Clinical features: (with repeated sun exposure and occurs after minor trauma) 1- Painless blisters, increased skin fragility on dorsal surfaces of hands 2- Facial hypertrichosis 3- hyperpigmentation. Diagnosis: is confirmed with pinkish-red fluorescence in the urine when examined with a Wood lamp (↑ urinary porphyrin). Treatment: Phlebotomy or Hydroxychloroquine may provide relief	Because of Vitamin B3 (niacin) deficiency Severe deficiency leads to Pellagra : 3 D's of B3 • Diarrhea, Dementia, Dermatitis : - Description: Skin of sun-exposed limbs becomes Indurated, lichenified, rough covered by dark scales & crusts - Distribution: is striking : 1- Butterfly region of the face 2- C3/C4 dermatome circumferential "broad collar" rash [Casal necklace] 3- Dorsa of hands and fingers ("gauntlet" of pellagra) 4- Dorsa of feet up to malleoli with sparing of the heel 

4- Chronic Photodamage

Actinic Keratosis

Actinic keratoses develop in genetically predisposed individuals 40-60 years of age under the influence of excessive chronic sun exposure. Distribution: Most commonly affected areas are face, ears, scalp and the dorsa of the arms and hands, also legs back & upper chest. Ligt microscopy, affected areas show: 1- Acanthosis (thickening of the epidermis) 2- Parakeratosis (retention of nuclei in the stratum corneum), 3- Dyskeratosis (abnormal keratinization) 4- Hyperkeratosis (thickening of stratum corneum). They are classically described as erythematous papules with a central scale due to hyperkeratosis → may become prominent and turn into "cutaneous horns". Rough "sandpaper-like" texture on palpation (better felt than seen) of affected areas is typical for this condition. (Their size does not exceed 10 mm) Course: Keratinocytes display various degrees of atypia. → Actinic keratosis is regarded as either a pre-malignant condition or a carcinoma in situ, but fewer than 1% of AKs will evolve into frank squamous cell carcinoma.

Skin Cancer

Squamous Cell Carcinoma



A large but subtle nodule, which is better felt than seen, on the vermilion border of the lower lip with areas of hyperkeratosis and erosion, arising in the setting of dermatoheliosis of the lip (cheilitis actinica).

• The most likely diagnosis of :

- 1- Asymptomatic (i.e., lesion-free),
- 2- In immunocompetent adult patient,
- 3- With a significant history of sun exposure,
- 4- with a **non-healing, isolated ulcer in the vermilion zone of the lower lip** → is **squamous cell carcinoma**

• Which is characterized by **invasive cords of POLYGONAL squamous cells** with **KERATIN pearls**.

• Risk factors:

- 1- **Exposure to sunlight "UVB"** (The single most important risk factor)
- 2- **Arsenic & aromatic hydrocarbons** are other well-known factors associated with SCC
- 3- **Actinic Keratosis**
- 4- **Significant history of alcohol & tobacco use :**
 - **SCC of the mucosa of the head and neck** is common in people with a significant history of alcohol & tobacco.
 - **First manifestation:** **Palpable cervical lymph node.**
 - **Best initial test is:** **Panendoscopy (triple endoscopy= esophagoscopy, bronchoscopy, laryngoscopy)**] to detect the primary tumor.

• Treatment:

- **Surgical excision:** **Mohs excision** (i.e., serial shallow excisions with histologic analysis to minimize cosmetic damage) may be performed for lesions on face;
- **Radiation** may be helpful in large lesions

Keratoacanthoma



- Formerly considered a pseudocancer it is now regarded by most as a **variant of SCC**
- **Rapidly growing "volcano-like" epithelial tumor** with potential for **tissue destruction** and (rare) metastasis.

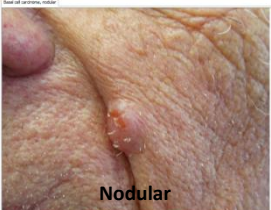
• Description: A **dome-shaped nodule with central hyperkeratotic plug**

• Treatment:

- Lesions are classically **self-limited**
- many are treated as **well-differentiated squamous cell carcinomas**.



Basal cell carcinoma



Nodular



Ulcerated



Pigmented



Superficial (scar-like)

- **BCC** is the **most common malignant tumor of the eyelid** and the **most common malignancy in mankind**

- It's **locally invasive, aggressive, and destructive** but **slow growing**, with **NO tendency to metastasize**.

• Risk factors :

- It usually occurs in **fair-skinned individuals** with a **history of prolonged sun exposure**.

• Description :

- **Clinically different types:** 1- **Nodular** 2- **Ulcerating** 3- **Pigmented** 4- **Sclerosing** 5- **Superficial**.
 - The lesions are most commonly **slow-growing nodules with a pearly quality and a rolled border**
 - There are often **telangiectasias** overlying and at the periphery of the lesion
 - **Bleeding and ulceration** are common features

- **Distribution:** (> 90% occur in the face / rarely found on the lips)

- Danger Sites : 1- **Medial & lateral canthi** 2- **Nasolabial fold** 3- **Behind the ears**

- Develops in Eyelid (Most common location for eyelid BCCs is the **lower eyelid margin**) → thinning or **loss of eyelashes** in the region of the tumor is typical.

*Spread from a periocular BCC into the orbit can occur and **enucleation of the eye** & **required to treatment**.*

• Histologically :

- BCC histologically characterized by **invasive clusters of spindle cells** surrounded by **palisaded basal cells**.

• Treatment: most appropriate treatment :

- **Surgical excision using microscopically-controlled margins** (**Mohs technique**).

• Differential Diagnosis of Eyelid BCC : **Chalazion**

- This is a **chronic granulomatous** condition that develops when a meibomian gland becomes obstructed.
- **Chalazion** initially presents as a **painful swelling that progresses to a nodular rubbery lesion**.
- Persistent or **recurrent chalazion** may be due to **meibomian gland carcinoma** (sebaceous carcinoma)
- **Basal cell carcinoma** frequently presents as a **solitary nodule on the lid margin** and may initially be clinically difficult to distinguish from a **chalazions**. → Patient therefore requires **histopathologic examination to rule out malignancy**.

Melanoma

- Melanoma is now the **most common malignancy in women aged 25 - 29 years old**.
- It is **second only to thyroid and breast cancer** in the age groups flanking 25 - 29 years.

• Risk factors:

- Fair skin types - History of blistering sunburns - Family history of melanoma
- Dysplastic nevus syndrome - Atypical nevi & greater than 100 typical nevi.
- **A recently changed mole is the strongest risk factor for malignancy;**

• Complaints: Patients often present complaining of:

- **mole that has changed in size or color (darkening or lightening)** or
- **mole that has become symptomatic (pruritic, painful or bleeding)**

• Clinical features:

- Distribution:

- Melanoma occurs as a **solitary lesion**, and **can occur anywhere on the skin**.
- Most common location → in men: **Back** / in women: **Legs**

- Description: A. B. C. D. Es of melanoma help in screening and early detection.

Clinical features of melanoma (ABCDEs)

- **Asymmetry:** When bisected, the 2 sides are not identical
- **Border irregularities:** Uneven edges, pigment fading off
- **Color variegation:** Variable mixtures of brown, tan, black & red
- **Diameter:** ≥6 mm
- **Evolving:** Lesion changing in size, shape, or color; new lesion

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• Types :

- 1- **Superficial spreading:** most common type; grows laterally before invasive growth occurs
- 2- **Nodular:** grows only vertically and becomes invasive rapidly; difficult to detect
- 3- **Acral lentiginous:** involves palms, soles, and nail beds
- 4- **Lentigo maligna:** long-lasting in situ stage before vertical growth

• Management :

- **The first step** is to confirm diagnosis histopathologically by **excisional biopsy** (**removing entire lesion** with **narrow margins** and **depth through S.C fat**)

Excisional biopsy with narrow margins is preferred because this allows confirmation of the diagnosis as:

- 1- Pigmented BCC
- 2- Some seborrheic keratoses
- 3- Atypical nevi → **can mimic melanoma clinically.**

Additionally, a **complete excision** allows determination of : **tumor depth (Breslow depth)**, **ulceration**, **presence of mitosis**, **regression**, **lymphatic and vascular involvement and host response.**

Excision with wider margins is **NOT recommended** until the diagnosis is confirmed as:

- 1- it would be inappropriate to remove margins around a benign lesion, and
- 2- because this may disrupt afferent cutaneous lymph flow and the ability to identify sentinel nodes.

• Primary Prevention :

- **Incidence** of malignant melanoma has been increasing dramatically → The risk factors for developing melanoma are both:

- 1- **Environmental:** **Excessive sun exposure**, tanning beds, PUVA therapy, multiple typical/congenital nevi, immunosuppression
- 2- **Genetic :** **Caucasian** and having a fair complexion, **blond hair** and **blue eyes**. / Personal history & family history.

- **Reducing sun exposure** is therefore the **best way** to prevent the development of melanoma, and

- **Use of protective clothing** (e.g. long sleeved shirts. wide-brimmed hats. sunglasses. tightly woven fabrics) is one of the **most effective methods**.

- **Regarding sunscreens:**

Sunscreens are useful adjuncts to photo-protection, but offer **insufficient protection from ultraviolet radiation (UVR) when used alone**.

Little to no protection against melanoma with the use of **sunscreen lotion with SPF 15 - 30** → However, there is evidence that SPF protects against **non-melanoma skin cancers such as squamous cell carcinoma**.

Overdependence on sunscreens may sometimes even increase or encourage outdoor exposure.

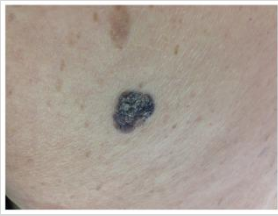
Sunscreens should be applied **15-200 min prior to sun exposure** to allow enough time for protective film development.



Benign Neoplasms & Hyperplasia

Seborrheic Keratosis

(Verruca Seborrhoica)



- Seborrheic keratosis is the **most common of the benign epithelial tumors** → more common with advancing age.
- Complaint:** Evolve over months to years
 - Are most commonly **asymptomatic** but can be **Pruritic or Tender**, particularly in locations that come into contact with clothing or jewelry.
- Clinical manifestations :**
 - The natural history is **slow enlargement** with increasing thickness.
- Distribution :**
 - Occur in many locations on the body but **tend to favor the face & trunk**.
 - Lesions **DO NOT occur on the palms & soles**.
- Description :** Lesions have a characteristic **waxy, "stuck on," warty, and well-circumscribed** appearance.
 - Early:** Small, 1- to 3-mm, barely elevated **Papule**, later a larger **Plaque** with or without pigment.
 - Late:** **Plaque** with **warty surface** and "stuck on" appearance.
 - Colors vary: from **pink/white** to **pale brown** to **dark brown** or **black**.
- Diagnosis:** is based mainly on clinical appearance. / Biopsy is rarely required in difficult cases.
- Differential Diagnosis:**
 - Tan Macules:** Early "flat" lesions may be confused with: 1- **Solar lentigo** or 2- **Spreading pigmented actinic keratosis**
 - Skin-Colored/Tan/Black Verrucous Papules/Plaques:** Larger pigmented lesions are easily mistaken for:
 - Pigmented basal cell carcinoma (BCC) or
 - Malignant melanoma (only biopsy will settle this)
 - Verruca vulgaris (but thrombosed capillaries are present in verrucae).
- Treatment:** No therapy is required unless 1- **Lesions become irritated** or 2- **Patient desire for cosmetic reasons**.
 - Treatment options: 1- Removal by snip/shave excision
 - 2- Cryosurgery
 - 3- Electrodesiccation.

Sebaceous Hyperplasia

- These are very common lesions in **older persons**
- Occurs in **solid organ transplant recipients treated with cyclosporine**.
- Description:**
 - Lesions are **1 to 3 mm** and have both **telangiectasia & central umbilication**
- Differential Diagnosis:** They are confused with **nodular BCCs**.
 - Two features distinguish sebaceous hyperplasia from nodular BCC:
 - sebaceous hyperplasia is soft to palpation, not firm as in nodular BCC and
 - with firm lateral compression it is often possible to elicit a very small globule of sebum in the valley of the umbilicated portion of the lesion.
- Treatment :**
 - Destroyed with light electrocautery**



Lipoma



- Lipomas are **single or multiple, benign subcutaneous tumors**.
- Description :**
 - Size: **Small BUT** may also **enlarge to > 6 cm**.
 - They are easily recognized because they are **soft, rounded, or lobulated and movable against the overlying skin**.
 - They occur especially on the **neck, trunk, extremities** BUT *can occur anywhere on the body*.
 - Dimple sign** : lateral compression with thumb and index finger produces a depression or "dimple"
- Histopathology :**
 - Lipomas are composed of **fat cells** that have the same morphology as normal fat cells **within a C.T framework**.
 - Angiolipomas** have a **vascular component** and may be **tender in** 1- **Cold ambient temperature** & 2- **with compression**.
- Treatment :**
 - Lipomas:** should be excised **only when considered disfiguring** :
 - Surgical excision
 - Liposuction can also be performed when liposomas are soft and thus have only a minor C.T component
 - Angiolipomas** often **require excision**

Vascular Malformation & Tumors

	Vascular Tumors (Hemangiomas)	Vascular Malformations
Presence at birth	Not present at birth but appear postnatally	The are errors of morphogenesis and are presumed to occur during intrauterine life → Most present at birth
Natural history	Phases: 1- Proliferating: grow rapidly during first year 2- involuting: slow during childhood over 2 – 6 years. 3- Involuting: by the age of 10 years.	Proportionate growth; can expand
Cellular	Endothelial hyperplasia	Normal endothelial turnover
Skeletal changes	- Occasional mass effect on adjacent bone; - rare hypertrophy	- Slow-flow: distortion, hypertrophy, or hyperplasia - Fast-flow: destruction, distortion, or hypertrophy

Vascular Tumors

Hemangioma of infancy

(Strawberry Hemangioma)



Involuting by age 5 years

- Most common tumor of infancy , with F : M ratio (3:1)

• Clinical Features :

Description:

- Soft bright Red (more violaceous when deeper), sharply demarcated, raised lesions.
- Appearing in first 2 months → rapidly expanding → then involuting by age 5–9 years

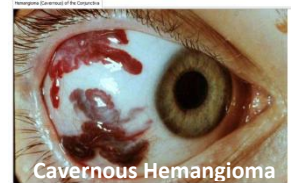
Distribution :

- Lesions are usually **solitary and localized** or **extend over an entire region** (figure) →
- **Head & neck 50% / trunk 25%**. Face, trunk, legs, oral mucous membrane.

• Special Presentations :

Deep Hemangioma (Formerly, Cavernous Hemangioma)

- Consist of **dilated vascular spaces** with **thin-walled endothelial cells**.
- **Presentation** as **soft blue, compressible masses** up to a few centimeters in size.
- Cavernous hemangiomas may appear on the **skin, viscera, deep tissues, mucosa** → If it involves the larynx : it can cause obstruction.
- They are **less likely to regress spontaneously** than capillary hemangiomas.
- Cavernous hemangiomas of 1- **Brain** & 2- **Viscera** → are associated with **von Hippel-Lindau disease**



Cavernous Hemangioma

• Treatment :

- **No intervention** is the best : *because spontaneous resolution gives the best cosmetic results*
- Treatment is indicated if hemangioma 1- Ulcerate 2- Obstruct vital structures 3- If life threatening as Hear Failure
 - **Surgical:** 1- Continuous wave or pulsed dye laser 2- Cryosurgery
 - **Medical:** 1- Intralesional and systemic high-dose glucocorticoids 2- Interferon α (IFN-α) 3- Propranolol.

Pyogenic Granuloma

- Very common **solitary eroded vascular nodule** that bleeds **spontaneously** or after minor trauma.
- Appears as a **bright red, dusky red, violaceous, or brown-black papule** with a collar of hyperplastic epidermis at the base



Vascular Malformations

Capillary Malformation(CM)

Port-Wine Stain

(Nevus flammeus)



• Clinical Features:

- It's **irregularly shaped, red or violaceous, macular CM** that is **present at birth** and **never disappears spontaneously** → tends to increase in proportion to size of the child → becomes raised & nodular causing significant disfigurement.

Distribution:

- Most commonly **involve the face** in the **distribution of the trigeminal nerve**, usually the superior & middle branches
- **Mucosal involvement of conjunctiva & mouth** may occur

• Syndromic CM :

Sturge-Weber syndrome (SWS) is the association of **Port-Wine Stain in the trigeminal distribution** with :

- 1- **Eye:** Vascular malformations in the eye AND
- 2- **Brain:** Leptomeninges & superficial calcifications of the brain

Klippel-Trénaunay-Weber syndrome

- May have an associated PWS overlying the deeper vascular malformation of soft tissue & bone.

Spider Angioma

(Spider Nevus)

- It consist of a **bright red central papule (dilated central arteriole)** surrounded by **telangiectatic network of dilated capillaries**.

- It's associated with **hyperestrogenic states**, such as : pregnancy, in patients receiving estrogen therapy, e.g., oral contraceptives, or in those with hepatocellular disease.
- Spider angioma may regress spontaneously.



Cherry Angioma

(Cherry Hemangiomas)
Senile Hemangiomas

- It appears during **third or fourth decade of life**.
- They **do not regress spontaneously** and, because **their number often increases with age**.
- They are always **cutaneous** and **NOT found on the mucosa or deep tissues**.
- **Description :**
- **sharply circumscribed areas of congested capillaries & post-capillary venules** in papillary dermis.



Drug-Induced Disorders

Immunologically Mediated Adverse Cutaneous Drug Reactions

Type I	Type II	Type III	Type IV
IgE-mediated ; immediate-type immunologic reactions: - Some patients can form drug-specific (IgE) on exposure to a medication → upon drug re-exposure IgE binds to basophils & mast cells → Symptoms. 1- Beta lactam drugs 2- Neuromuscular blocking agents 3- Quinolones 4- Platinum-containing chemotherapeutics	Drug + cytotoxic antibodies cause lysis of cells such as platelets or leukocytes 1- Penicillin 2- Sulfonamides 3- Quinidine 4- Isoniazid	IgG or IgM antibodies formed to drug; immune complexes deposited in small vessels activate complement and recruitment of granulocytes 1- Immunoglobulins 2- Antibiotics 3- Rituximab 4- Infliximab	Cell-mediated immune reaction ; sensitized lymphocytes react with drug, liberating cytokines, which trigger cutaneous inflammatory response 1- Sulfamethoxazole, 2- Anticonvulsants 3- Allopurinol
Urticaria/Angioedema - Onset is rapid (seconds to minutes) - Symptoms can range from : -- Mild : urticaria, pruritus, flushing -- Severe : - Angioedema of larynx - Anaphylaxis Mild manifestations of a drug allergy are usually treated with : 1- Antihistamines and 2- Discontinuation of offending drug.	1- Petechiae due to : Thrombocytopenic Purpura 2- Drug-induced pemphigus	1- Vasculitis 2- Urticaria 3- Serum sickness : - 5 to 21d after initial exposure: Minor form : fever, urticaria, arthralgia Major (complete) form : - Fever, urticaria, angioedema, - arthralgia, arthritis, - lymphadenopathy, - eosinophilia, ± endocarditis, ± nephritis.	1- Morbilliform exanthematous reactions 2- Fixed drug eruption 3- lichenoid eruptions 4- Stevens-Johnson syndrome 5- Toxic epidermal necrolysis

Stevens Johnson syndrome (SJS) & Toxic Epidermal Necrolysis (TEN)

- **SJS and TEN** are acute life-threatening mucocutaneous reaction characterized by **extensive necrosis and detachment of the epidermis**.
- Both are severe variants of an **identical pathologic process** and **differ only in the percentage of body surface involved** :
 - **SJS**: <10% epidermal detachment
 - **SJS/ TEN overlap**: 10–30% epidermal detachment.
 - **TEN**: >30% epidermal detachment.
- Now consensus that **SJS and TEN** are **different from Erythema Multiforme (EM)**.
- **Pathogenesis**:
 - Drugs or their metabolites act as haptens and render keratinocytes antigenic by binding to their surfaces → cytotoxic immune reaction aimed at the destruction of keratinocytes expressing foreign (drug-related) antigens.
 - Epidermal injury is based on the **induction of apoptosis**.
- **Clinical Manifestations**: 4 – 28 day after exposure to trigger (2 days after repeat exposure).
 - **Prodromes** :
Acute-influenza like (fever , malaise , arthralgias) **1 – 3 days** before mucocutaneous lesions.
 - **Skin lesions** :
 - 1) **Prodromal Rash** : Morbilliform, can be **target lesion-like** eruption
 - 2) **Necrosis & sloughing of epidermis** : (exfoliation of the skin)
 - First appears as **macular** areas with crinkled surface that enlarge and coalesce.
 - **Sheetlike loss of epidermis**.
 - **Raised flaccid blisters that spread with lateral pressure (Nikolsky sign)** on erythematous areas.
 - **Mucous membranes** :
There is typically a sudden onset of **mucocutaneous** lesion over two sites usually:
 - 1) **Oral** : Erythema , painful erosions & blisters
 - 2) **Conjunctival** : 85% have conjunctival lesions
Hyperemia, pseudomembrane formation; keratitis, corneal erosions; later synechiae between eyelids and bulbar conjunctiva.
- **General Findings**:
 - **Fever , mentally alert**. Distress due to severe pain.
 - **Cardiovascular** : pulse may be >120 beats/ min. Blood pressure.
 - **Renal**: tubular necrosis may occur. Acute renal failure.

Stevens-Johnson syndrome & toxic epidermal necrolysis	
Nomenclature	<10% of BSA: SJS 10%-30%: SJS/TEN overlap >30%: TEN
Clinical features	• 4-28 days after exposure to trigger (2 days after repeat exposure) • Acute influenza-like prodrome • Rapid-onset erythematous macules, vesicles, bullae • Necrosis & sloughing of epidermis • Mucosal involvement
Common triggers	Drugs • Allopurinol • Antibiotics (eg, sulfonamides) • Anticonvulsants (eg, carbamazepine, lamotrigine, phenytoin) • NSAIDs (eg, piroxicam) • Sulfasalazine Other • <i>Mycoplasma pneumoniae</i> • Vaccination • Graft-vs-host disease

BSA = body surface area; NSAIDs = nonsteroidal anti-inflammatory drugs; SJS = Stevens-Johnson syndrome; TEN = toxic epidermal necrolysis.



Erythema Multiforme

- A common **reaction pattern** (to a variety of antigenic stimuli) of **blood vessels in the dermis** with **secondary epidermal changes**.
- Reaction is most commonly to **Herpes Simplex Virus (HSV) Infection**.

Antigenic stimuli

Infection Especially following herpes simplex, *Mycoplasma* .

Drugs Sulfonamides, phenytoin, barbiturates, phenylbutazone, penicillin, allopurinol.

Idiopathic Probably also due to undetected herpes simplex or *Mycoplasma*.

Clinical Features :

Section 7.)

- Description :

- Dull red **iris or target-like lesions** (sharply defined flat papules with a central vesicle) are typical →

- Distribution: Bilateral and often symmetrical

- Either : 1- Localized to hands & face or
- 2- Generalized Bilateral and often symmetric.

Dorsa of hands, palms, and soles / forearms / feet / face / elbows and knees / penis and vulva

Mucous Membranes: Erosions with fibrin membranes; occasionally ulcerations:

- lips, oropharynx, / - Nasal / Vulvar, anal.
- Eyes: with corneal ulcers, anterior uveitis.

Complications of HSV Infections

- **Eczema herpeticum** : Usually follows autoinoculation of HSV (most commonly orolabial herpes) to **atopic dermatitis**.
- **S. aureus superinfection**: Occurs with eczema herpeticum.
- **Erythema multiforme** : In some individuals **with recurrent HSV infections**, erythema multiforme may occur with each recurrence



Mild Forms (EM Minor)	Severe Forms (EM Major)
	Most often occurs as a drug reaction
- Vesicles BUT: - NO bullae Eruption usually confined to extremities, face	- Lesions are severe, extensive, - Tendency to become confluent & bullous, - Positive Nikolsky sign in erythematous lesions
- Little or NO mucous membrane involvement	- Always with mucous membrane involvement
- NO systemic symptoms. Recurrent EM minor is usually associated with an outbreak of herpes simplex (HSV) preceding it by several days.	- Systemic symptoms: fever, prostration. - Cheilitis and stomatitis interfere with eating; - vulvitis and balanitis with micturition. - Conjunctivitis can lead to keratitis - Lesions also in pharynx and larynx.

Differential Diagnosis

Erythroderma (Exfoliative Dermatitis)	• It's a serious, life-threatening, reaction pattern of the skin characterized by: generalized and uniform REDNESS and SCALING involving practically the entire skin. • 50% of patients have history of preexisting dermatosis : in the order of frequency: <i>Psoriasis, atopic dermatitis, adverse cutaneous drug reactions, cutaneous T cell lymphoma, allergic contact dermatitis, and pityriasis rubra pilaris</i> • It is associated with fever, malaise, shivers, and generalized lymphadenopathy • There is a loss of scalp and body hair, and nails separated from nail bed (onycholysis) • There may be hyperpigmentation in whose normal skin color is brown or black.	
Erythema Induratum (Nodular Vasculitis)	• Nodular vasculitis is a form of lobular panniculitis associated with subcutaneous blood vessel vasculitis with subsequent ischemic changes that produce lipocyte injury, necrosis, inflammation, and granulation. • Appears as a nodular eruption in patients with Tuberculosis. • On examination: There are crops of small, tender, erythematous nodules involving the shins and calves → may undergo necrosis forming slowly healing ulcers.	
Erythema Nodosum	• The most common type of panniculitis: - Panniculitis is the term used to describe diseases where the major focus of inflammation is in the S.C tissue • EN is an important and common acute inflammatory/ immunologic reaction pattern of the subcutaneous fat. • Characterized by : Appearance of painful nodules on the lower legs. Lesions are bright red and flat but nodular upon palpation. • EN is not a disease but a cutaneous reaction pattern to various etiologic agents.	

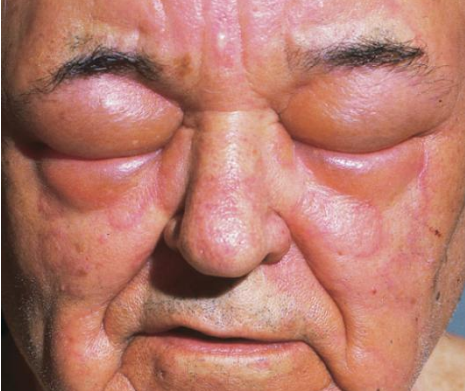
Warfarin-induced skin necrosis :

- It's a serious complication of **oral anticoagulants**.
- **Protein C deficiency** is sometimes associated with this condition.
- **Females** are most commonly affected.
- **Clinical Features:**
 - **Distribution:**
 - The commonly involved sites: **breasts, buttocks, thighs, and abdomen**.
 - **Description:**
 - The initial complaint is **pain**, followed by → **bullae formation & skin necrosis** (It mostly occurs within weeks after starting therapy)
- **Treatment :**
 - **Vitamin K** should be promptly administered in the *early stages of the lesion*
 - **Warfarin is discontinued** if the lesion progresses & **Heparin** should be used to *maintain anticoagulation until necrotic lesions heal*.
 - **Skin grafting:** Few patients require it.

Urticaria & Angioedema

- **Urticaria :**
 - Urticaria is composed of **wheals** (transient edematous papules and plaques, usually pruritic and due to edema of the papillary body) .
 - The wheals are **superficial, well defined**.
- **Angioedema :**
 - Angioedema is a **larger edematous area** that involves the **dermis and subcutaneous tissue**.
 - It is **deep** and **ill defined**.

THUS, Urticaria and angioedema are **the same edematous process** BUT involving different levels of the cutaneous vascular plexus.

		
Acute urticaria		Acute urticaria & angioedema
- Acute onset and recurring over <30 days. - Usually large wheals often associated with angioedema - Etiology - Often IgE-dependent with atopic diathesis; - Related to foods, parasites, and drugs (penicillin). - Description : - Sharply defined round/oval wheals, small (<1 cm) to large (>8 cm), erythematous. - Clearing of the central region may occur (white with an erythematous rim). - Lesions may coalesce, producing an annular, serpiginous pattern. Pruritus → In angioedema of palms and soles pain.		Note that there are both: 1- Superficial wheals & 2- Deep, diffuse edema. Angioedema : Skin-colored, transient enlargement of portion of face, eyelids, lips, tongue, glottis & larynx , or other sites due to subcutaneous edema Laryngeal edema can cause airway obstruction and be life threatening.

ACE inhibitors + Angioedema

- ACE inhibitors are the most common cause of **acquired angioedema**.
- **Mechanism :**
 - **ACE** is also known as **kininase** , it functions to degrade bradykinin.
 - When ACE is inhibited → levels of **bradykinin increase** → thereby leading to **angioedema**.
Angioedema occurs due to the pro-inflammatory action of bradykinin, which promotes edema, inflammation and the sensation of pain.
 - It is important to note that angioedema from ACE inhibitors can occur **at ANYTIME**, not just within weeks of starting the medication.
- **Treatment:** (ACE-inhibitor should be stopped immediately)
 - First step in management of angioedema is : **to check for 1- Airway compromise & 2- Vasomotor instability**, → which require **S.C epinephrine administration if present**.
 - If airway obstruction fails to respond to epinephrine → **emergency tracheostomy** is done

Hereditary Angioedema (HAE)

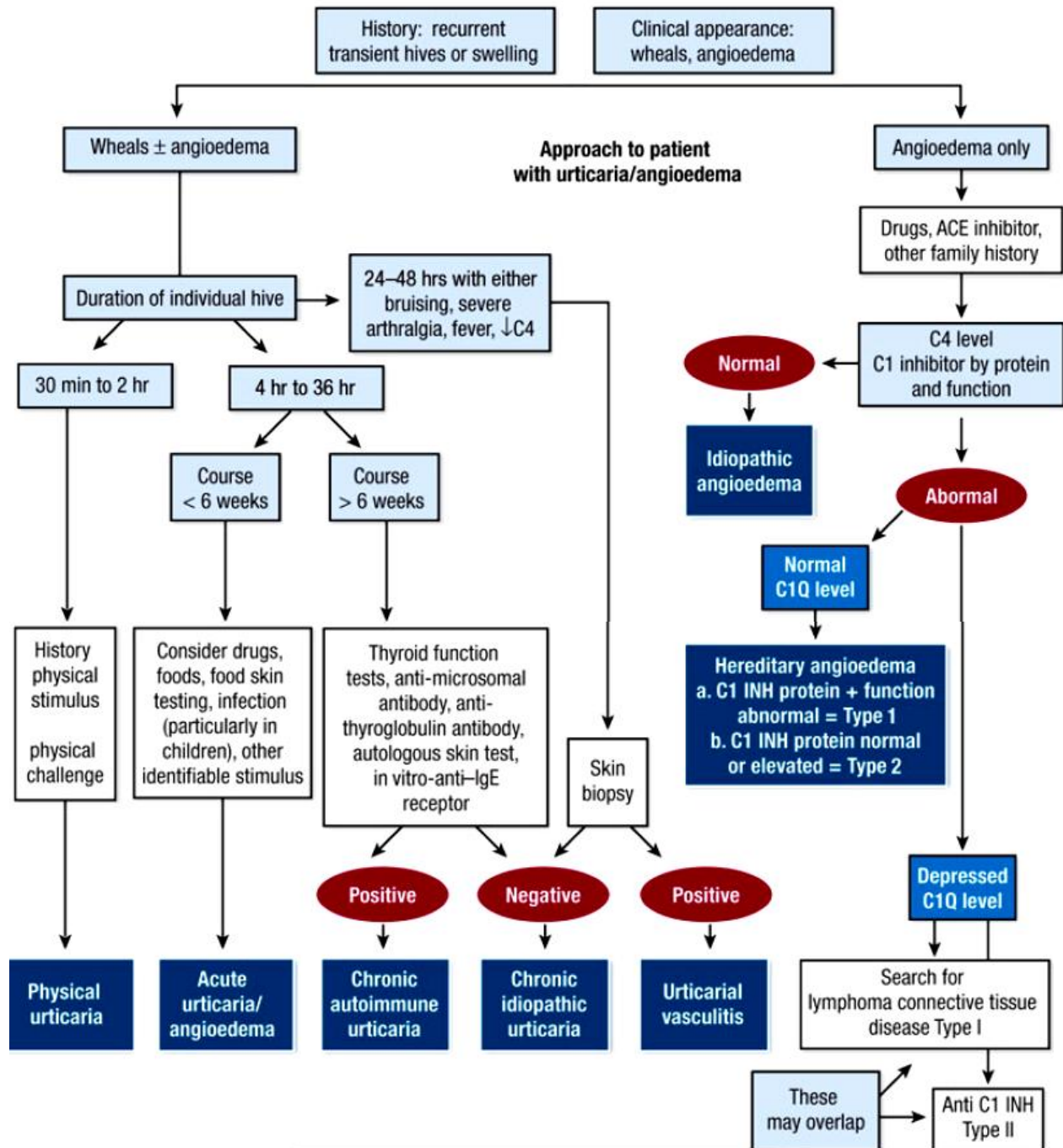
- A serious **autosomal dominant disorder**; may follow trauma (physical and emotional).
- **Pathophysiology:**
 - **C1- esterase inhibitor** deficiency, dysfunction or destruction

Since C1-esterase inhibitor is also the major inhibitor of the **Hageman factor & kallikrein**, the two enzymes required for **kinin formation** → elevated levels of the **edema-producing factors C2b & bradykinin**

 - Episodes usually **follow an infection, dental procedure or trauma.**
- **Clinical Manifestations :**
 - Usually presents in late childhood
 - It is characterized by a rapid onset of the following symptoms :
 - (1) non-inflammatory **edema of the face, limbs, genitalia**,
 - (2) **laryngeal edema**, and
 - (3) **edema of the bowels resulting in colicky abdominal pain.**
- **Laboratory Abnormalities :**
 - **C1- esterase inhibitor** : **Decreased** levels of (85%) / dysfunctional inhibitor (15%),
 - **C1q levels** : **normal** in hereditary angioedema (depressed in acquired forms)
 - **C4 levels** : are depressed in all forms of angioedema.

Clinical features of acute urticaria	
Clinical presentation	• Well-circumscribed, raised erythematous plaques • Lesions can be oval, round, or serpiginous up to several centimeters in diameter • Intense pruritus • Lesions can worsen over minutes to hours, then resolve within 24 hours
Etiologies	• Infections (viral, bacterial, parasitic) • IgE mediated (antibiotics, insect bites, latex, food, blood products) • Direct mast cell activation (narcotics, muscle relaxers, radiocontrast medium) • NSAIDs • Idiopathic (up to 50% of patients)

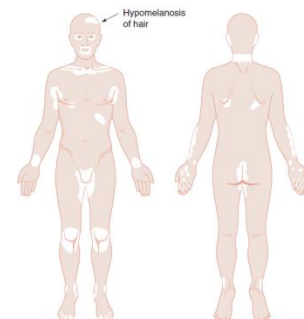
NSAID = nonsteroidal antiinflammatory drug.



Pigmentary Disorders

Vitiligo

- It's a **depigmenting disorder** due to loss of epidermal melanocytes.
- Pathogenesis:** it's thought to be an autoimmune process which causes **destruction of the melanocytes**.
progressive, acquired, chalk-white, bilateral (usually symmetric),
- Associations:**
Vitiligo is associated with other autoimmune conditions like: **pernicious anemia, autoimmune thyroid disease** (usually Graves' or chronic autoimmune thyroiditis), **type 1 diabetes mellitus, primary adrenal insufficiency, hypopituitarism, and alopecia areata**
- Clinical Features :**
 - Description:** **progressive, acquired, chalk-white, bilateral (usually symmetric), sharply marginated macules with hyperpigmented borders** in typical sites.
 - Distribution:**
 - 1) Localised type :** is characterized by one or several macules in a single site;
 - 2) Generalized vitiligo**
 - is more common and is characterized by **widespread distribution of depigmented macules**, often in a **remarkable symmetry**
 - Typical macules occur **around the eyes and mouth and on digits, elbows, and knees, as well as on the low back and in genital areas.**
- Management :** - *Spontaneous repigmentation may occur.*
 - 1- Sunscreens :** All patients need sunscreens to protect the depigmented skin from sun damage.
 - 2- Repigmentation :**
 - Localized vitiligo :** may be treated with topical :
 - Topical corticosteroids (not used on face due to glaucoma & atrophy) so we alternate with :
 - Topical calcineurin inhibitors (immunomodulators) **Tacrolimus** and **pimecrolimus**
 - Topical photochemotherapy
 - Generalized vitiligo :**
 - Systemic photochemotherapy (oral PUVA)
 - Narrow-band ultraviolet light B (UVB, 311 nm) is now the treatment of choice
 - Excimer laser (308 nm): Produces best results in the face.
 - 3- Depigmentation :**
Bleaching: Bleaching of *normally pigmented skin* with **monobenzylether of hydroquinone (MEH)** cream is a permanent, irreversible process.



Differential Diagnosis

Hypopigmented lesions

- 1- Vitiligo**
- 2- Leprosy**
- 3- Pityriasis alba.**
 - Cause: unknown
 - Mostly : seen on the face of children in summer in temperate climates.
 - It is a white area (alba) with very mild scaling (pityriasis).
 - Relatively indistinct margin under Wood light
 - It is self-limited.
- 4- Pityriasis versicolor**
 - Common in adults, on trunk, covered with fine scales, Wood light shows golden yellow fluorescence and confirmed with LM examination.
- 5- Albinism**
- 6- Nevus depigmentosus**
- 7- Post inflammatory**
- 8- Chemical leukoderma**

Alopecia

Alopecia Areata

- A localized loss of hair in round or oval areas with **NO apparent inflammation of the skin**
- Pathogenesis :**
 - Follicular damage occurs in **anagen** followed by → rapid transformation to **catagen** and to → **telogen**; then to → **dystrophic anagen status**.
 - While the disease is active, follicles unable to progress beyond early anagen and do not develop normal hair.
- Clinical Features :**
 - Description :** Skin is Normal
 - Round patches of hair loss.**
 - Single or multiple. May coalesce.
 - It's often **sharply defined**.
 - Normal-appearing skin** with follicular openings present .
 - "Exclamation mark" hairs :** Diagnostic broken- off stubby hairs (distal ends are broader than proximal ends) seen at margins of hair loss areas.
 - Distribution:**
 - Most commonly on scalp - Any hair-bearing area : Beard, eyebrows, eyelashes, pubic hair.
- Management :** *No curative treatment is currently available. Treatment for AA is unsatisfactory.*
Treatment directed at inflammatory infiltrate and growth inhibitor factors produced by inflammation.
 - 1- Glucocorticoids :** Glucocorticoids *Topical / Intralesional Injection / Systemic Glucocorticoids :*
 - 2- Systemic Cyclosporine** Induces regrowth, but AA recurs when drug is discontinued.
 - 3- Induction of Allergic Contact Dermatitis** - By Dinitrochlorobenzene (DNCP) , but local discomfort.
 - 4- Oral PUVA (Photochemotherapy) :** Entire body must be exposed



Inflammatory Disorders

Psoriasis

- It's chronic genetically determined, inflammatory and hyperproliferative skin disease.

- Classification :**

- Psoriasis vulgaris:**
 - Acute guttate
 - Chronic stable plaque
- Psoriatic erythroderma**
- Pustular psoriasis**
- Psoriasis arthritis**

- Triggering Factors :**

- External triggering factors :**

- Physical trauma (Koebner phenomenon)**
rubbing & scratching stimulate the psoriatic proliferative process.
Occurs 7 to 14 days after injury

- Systemic triggering factors :**

- Infections :** acute strept. infection precipitates *guttate psoriasis*.

- Clinical Features :**

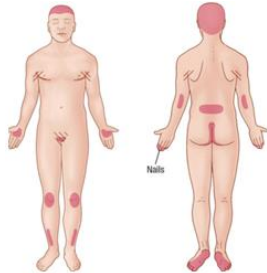
- Description :**

The classic lesion of psoriasis is:

- Sharply margined, raised red **plaque** with a white scaly surface.
- Lesions can vary in size from **papules**, which may grow to **plaques**, that coalesce to form geographic lesions.
- Scales are lamellar, loose, and easily removed by scratching.
- Removal of scale results in the appearance of minute blood droplets (**Auspitz sign**).

- Distribution:** (Single lesion or lesions)

- Extensor surfaces: elbows, knees,
- Scalp, Most obvious at the hair line & behind the ears.
- palm/soles
- intergluteal cleft,



- Treatment :**

- All patients should use **emollients**, such as Eucerin, Lubriderm, Aquaphor, and Vaseline or mineral oil.
- Salicylic acid** is used to remove heaped up collections of scaly material so the other therapies can make contact.
- If the disease is relatively localized, **topical steroids** are used.
- Severe disease also needs **coal tar or anthralin derivatives**.
- To avoid the long-term use of steroids, which can cause skin atrophy, and to avoid coal tars, which are messy, one can substitute:
 - topical vit. D** → calcipotriene
 - Topical Vit. A** → Tazarotene
- When > 30 percent of the body surface area is involved :**
 - it is difficult to use topical therapy routinely to control disease.
 - Such patients can be treated with **ultraviolet light**. *This is the most rapid way to control extensive disease.*
- The most severe, widespread, and progressive forms of the disease can be controlled with **methotrexate**. However, this therapy has the highest toxicity and may cause **liver fibrosis**.
- The newest therapies are **immunomodulatory biological agents**, such as
 - Alefacept**
 - Efalizumab**
 - Etanercept**
 - Infliximab**.



Lichen Planus

- It is an acute or chronic inflammatory dermatosis involving skin and/or mucous membranes.

- Clinical features:**

Onset : Acute (days) or insidious (over weeks).

Duration : Lesions last months to years,

Symptoms :

- Asymptomatic or pruritic .
- Mucous membrane lesions are painful, especially when ulcerated.

Description :

Characterized by **flat-topped**, pink to violaceous, with white lines (Wickham striae), shiny, pruritic, polygonal **PAPULES**.

The features of the lesions have been designated as the four P's :

Papule, **P**urple, **P**olygonal, **P**ruritic.

- Grouped, annular, or disseminated scattered discrete lesions when generalized .
- Koebner phenomenon:
May present in a linear arrangement after trauma.
- In dark-skinned individuals, **postinflammatory hyperpigmentation**.

Distribution:

- Mucous membranes of the cheeks & lips .
- Common on the flexor surfaces of wrists, the forearm & legs.
- lumbar region, glans penis
- shins (thicker, hyperkeratotic lesions),
- Scalp.



- Treatment :**

1) Local Therapy :

- Glucocorticoids :**
 - Topical WITH occlusion** for cutaneous lesions.
 - Intralesional** is helpful for :
 - Symptomatic cutaneous or
 - Oral mucosal lesions and lips.
- Cyclosporine and Tacrolimus Solutions :**
Retention "mouthwash" for severely symptomatic oral LP.

2) Systemic Therapy :

- Glucocorticoids**
- Cyclosporine:** In very resistant and generalized cases,
- Systemic Retinoids (Acitretin)** (1 mg/kg /day).
is helpful as adjunctive measure in severe (oral, hypertrophic) cases.

3) PUVA Photochemotherapy :

In individuals with generalized LP or cases resistant to topical therapy.

Important Skin conditions

Acanthosis Nigricans (AN)

- A cutaneous marker related to hereditary, obesity, endocrine disorders (particularly diabetes), drug administration & malignancy.
- **Clinical features:**
 - **Description:** - hyperkeratotic, hyperpigmented plaques with a classic "velvety" texture.
 - **Skin tags (acrochordons)**, which are pedunculated outgrowths of normal skin, are also commonly present on regions affected by AN → figure
 - **Distribution:** Flexural areas : axilla, groin, posterior neck, are the most common locations affected.
- Depending on the etiology, AN can be divided into benign and malignant forms :



Benign AN

- It's typically seen in younger individuals.
- It is associated with insulin-resistant states: (eg, diabetes mellitus, obesity, polycystic ovarian syndrome)
Increased levels of insulin and/or insulin-like growth factors are thought to stimulate epidermal and dermal proliferation.

Malignant AN

- The sudden appearance of such skin changes in middle-aged or elderly patients is suggestive of underlying malignancy.
- It is associated with underlying neoplasms, especially of the gastrointestinal and genitourinary tracts.
- These patients are NOT obese (but instead may have lost weight)
- Lesions can occur in uncommon areas (eg, mucous membranes, palms, soles).

Miscellaneous Disorders

Ichthyosis Vulgaris

Ichthyosis: History of normal skin at birth, with gradual progression to dry scaly skin, is typical of ichthyosis.

- This condition can be hereditary or acquired.
- The skin is usually dry and rough with horny plates over the extensor surfaces of the limbs.
In children, there may be relative sparing of the face and diaper area.
- The condition worsens in the winter because of increased dryness, and sometimes referred to as "lizard skin."



Skin conditions & associated diseases	
Skin conditions	Important associated conditions
• Acanthosis nigricans	• Insulin resistance • Gastrointestinal malignancy
• Multiple skin tags	• Insulin resistance • Pregnancy • Crohn disease (perianal)
• Porphyria cutanea tarda • Cutaneous leukocytoclastic vasculitis (palpable purpura) secondary to cryoglobulinemia	• Hepatitis C
• Dermatitis herpetiformis	• Celiac disease
• Sudden-onset severe psoriasis • Recurrent herpes zoster • Disseminated molluscum contagiosum	• HIV infection
• Severe seborrheic dermatitis	• HIV infection • Parkinson disease
• Explosive onset of multiple itchy seborrheic keratoses	• Gastrointestinal malignancy
• Pyoderma gangrenosum	• Inflammatory bowel disease

